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Spinal Puncture Headache

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• *Headache is the commonest complication of spinal puncture. There is no significant difference in the incidence of headache after lumbar puncture, whether or not the puncture is followed by injection of an anesthetic agent. The sequence of events leading to postlumbar puncture headaches is probably (1) decreased volume of cerebrospinal fluid with lowered pressure; (2) increased differential between the pressure of the cerebrospinal fluid and the intracranial venous pressure; (3) dilation of venous structures with increase in brain volume; and (4) production of tension on the pain sensitive areas in the cranium.*

Prevention of postlumbar puncture headache consists largely in attempts to avoid the development of the pressure differential between that of the cerebrospinal fluid and intracranial venous pressure. Treatment consists of analgesics, hydration and attempts to restore normal cerebrospinal fluid pressure.

HEADACHE is probably the most common untoward complication of spinal puncture. August Bier suffered a severe headache following his submission to the first attempt to produce spinal anesthesia in man in 1898. The incidence of headache following spinal puncture seems to vary little whether or not the puncture is followed by the injection of an anesthetic agent. Babcock,[5] in 1913, reported an incidence of headache of 21 per cent in 5,000 cases. Koster and Weintrob,[25] in 1930, reported postspinal puncture headache in 10 per cent of 6,000 patients who received spinal anesthesia. Woodbridge,[43] in 1937, reported a 4 per cent incidence of spinal puncture headache in 1,381 patients. Jennings[21] reported 30.6 per cent in 1939, while Hingson, Ferguson and Palmer,[19] in 1943, reported an incidence of only 1 per cent in 5,150 cases. Although there is wide variation in the reported incidence of headache following spinal puncture, the majority of the recent reports indicate that the incidence is probably between 10 and 20 per cent.[2, 4, 10, 12, 15, 39, 40]

Recently the authors made a study of a series of 515 consecutive cases in which spinal anesthesia was employed, with the idea of determining the incidence of spinal puncture headache. No attempt was made to direct the patient's attention away from the possibility of headache following anesthesia. In fact, each patient was told that headache was a common sequel of spinal puncture. Each was then asked specifically if he did have a headache after operation. Even though the question was "leading," the answers obtained indicated that the incidence was almost the same as that commonly reported. Furthermore, contrary to a previous assumption, early ambulation after operation apparently did not materially increase the number of postspinal puncture headaches. There seemed to be an appreciably greater incidence of postpunc-

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TABLE 1.—*Headache following spinal anesthesia*

Type	Number	Postspinal headaches		Other headaches		All headaches	
		Number	Per cent	Number	Per cent	Number	Per cent
Spinal	291	17	5.8	8	2.7	25	8.6
Continuous spinal	224	21	9.4	10	4.5	31	13.9
Total	515	38	7.4	18	3.5	56	10.8

ture headaches, however, when the agent was administered by the continuous or fractional technique (Table 1).

Not all headaches complained of following a spinal puncture can be considered to be postpuncture headaches. Headaches unaffected by postural changes and described by the patient as similar to those they have been subject to were not considered to be true postspinal puncture headaches. In the present series the final judgment as to whether the discomfort was a new development or merely the recurrence of a chronic malady was left to the patient.

For many years postspinal puncture headache has been considered to be related to changes in cerebrospinal hydrodynamics. Most observers have ascribed these headaches to lowered intracranial pressure,[2, 20, 28, 29, 33, 37, 42] although a few have noted that they may be associated with increased pressure.[17, 23]

With the patient in the horizontal position, intracranial (vertex) pressure is usually the same as lumbar and cisternal pressure. Intracranial pressure usually varies from 50 to 180 mm. of mercury. With the patient erect, lumbar pressure may be as high as 300 to 500 mm. of mercury while the intracranial pressure may drop to +40 or even to —85 mm. of pressure. On occasions it may even reach —300 mm. of mercury.[42] Changes in cerebrospinal fluid pressure probably closely parallel changes in venous pressure in the normal subject, although the cerebrospinal fluid pressure is usually somewhat higher than the venous pressure at any given level.

Wolff[42] demonstrated that there are certain sensitive structures in the cranium the stimulation of which will produce pain. The pain produced by the stimulation of these regions may be interpreted by the subject as headache. Demonstration of these pain-sensitive areas was carried out on human beings undergoing intracranial surgical procedures under local anesthesia. The principal structures found to be pain-sensitive are: (1) the great venous sinuses, (2) the venous tributaries to the sinuses, (3) parts of the dura near the base of the brain, (4) dural arteries and (5) cerebral arteries at the base of the brain. The afferent nerve pathways for pain from these areas are by way of the fifth nerve for all those structures above the tentorium, while subtentorial pain is transmitted largely through the ninth and tenth nerves. Some of the pain, low in the suboccipit, possibly is transmitted by the upper cervical nerves. Pain caused by stimulation of pain-sensitive areas above the tentorium is referred to the head anterior to the ears and in the region of the eyes, while pain posterior to the ears and in the suboccipit is probably from stimulation of sensitive areas below the tentorium.

Drainage headache is a well established clinical entity. The universal complaint of headache, usually very severe, during the course of encephalography under local anesthesia is evidence of the syndrome of drainage headache. It is a common observation that headache of this kind develops quite early in the procedure when only a small amount of fluid has been removed. Wolff[42] produced headache experimentally in 11 subjects by the drainage of cerebrospinal fluid and found that headache developed usually when about 20 cc. of fluid had been removed. The headache became worse as more fluid was withdrawn and could be relieved in all subjects by restoring the cerebrospinal fluid pressure to its previous value. These experimentally produced drainage headaches as well as the headache that is a sequel to encephalography are in every way comparable to the headache that may follow spinal anesthesia or diagnostic lumbar puncture. Wolff stated that headache probably will start when approximately 10 per cent of the estimated total volume of cerebrospinal fluid is removed.

Although the overwhelming majority of the evidence seems to favor the hypothesis that the most important factor in the production of postlumbar puncture headache is the lowering of the cerebrospinal fluid pressure, other possible contributing factors must be considered.

Irritation of the pia-arachnoid by the anesthetic agent has been suggested as a possible cause of postspinal anesthetic headache. This seems unlikely, considering the very high dilution of the agent in the cerebrospinal fluid and the usual lack of a significant rise in protein content and cell count of the cerebrospinal fluid following spinal anesthesia.[6, 8, 24] At operations involving opening of the dura following spinal anesthesia, seldom is any evidence of dural or meningeal irritation observed. Furthermore, the incidence of headache following diagnostic lumbar puncture is usually reported as high as that following spinal anesthesia.

Meningitis undoubtedly can cause severe headache, but only on very rare occasions could this be considered a cause of postpuncture headache. Other

symptoms of meningitis would probably be so evident as to leave little doubt as to whether this was the principal etiologic factor in a particular case.

Weintraub, Antine and Raphael[40] suggested that the decrease in intra-abdominal pressure after delivery is an important factor in the production of postpuncture headache when spinal anesthesia is used in obstetrics. They postulated that the pooling of the blood in the splanchnic vessels after the sudden release of intra-abdominal pressure lowers the pressure in the intracranial veins, permitting the brain to sag as some of its basilar cushion is lost. They advocated the use of tight abdominal binders as an aid in the correction of this imbalance in the intracranial circulation. They expressed belief that the increased return of blood following abdominal compression increases the pressure in the right auricle, which is transmitted by the jugular veins to the cerebral vessels. A possible additional mechanism whereby tight abdominal binders might contribute to restoration of proper balance to the intracranial circulation is suggested by the work of Batson.[7] He demonstrated that the current of the flow of blood in the vertebral plexus of veins can be reversed by increased intra-abdominal pressure.

Although there is experimental evidence to the effect that even pronounced increase in intracranial pressure usually does not cause headache,[42] certain clinical observations seem to indicate that at times increased intracranial pressure may be a factor in the production of headache. Hand[17] observed that some patients in whom headache developed incidental to repeated subarachnoid injections of ammonium sulfate for the relief of intractable pain had increased intracranial pressure. It is not uncommon for patients to complain of transitory headache occurring at the time of injection of the spinal anesthetic agent when large volumes (10 to 20 cc.), such as are used with the Howard-Jones technique for Nupercaine, are employed.

In spite of the fact that other factors may be present and at times contribute to postspinal puncture headache, it must be concluded that in most instances this complication is related to a lowering of the cerebrospinal fluid pressure owing to a reduced volume of fluid. Obviously, this decrease in cerebrospinal fluid volume could be caused by a decrease in the fluid output or by leakage of the fluid after it is formed. There seems little doubt that it is the result of leakage through the hole left in the dura by the spinal puncture needle. There is ample evidence that this hole remains for several days after spinal puncture.[2, 13, 32, 34] Mixter[32] noted that the hole made by the spinal needle in the dura was present at operation six days after spinal puncture. Franksson and Gordh[12] observed the hole still patent as late as 14 days after spinal puncture. The negative pressure reported to exist in the epidural space could be a factor contributing to the lowering of cerebrospinal fluid volume because of a hole in the dura. The incidence of failure in attempts to produce spinal anesthesia within the first few days after spinal puncture is notoriously high. In one case observed by the authors, three successive unsuccessful attempts were made to induce spinal anesthesia for the removal of a ruptured intervertebral disk. The anesthetic agent was given approximately 48 hours after myelography. Even though a free flow of cerebrospinal fluid was obtained with each attempt, no more than a few scattered areas of patchy anesthesia in the thighs and lower trunk could be induced. General anesthesia was induced, and at operation a large collection of epidural fluid was noted, and it was observed that the anterior and posterior walls of the dura were practically in apposition. Undoubtedly it was into this fluid-containing epidural space that the anesthetic agent was injected.

Further evidence that lowering of cerebrospinal fluid pressure is the prime etiologic factor in the production of postspinal puncture headache is the fact that measures which restore the volume of cerebrospinal fluid tend to relieve the headache. Injection of normal saline solution into the subarachnoid space is always followed by relief.[2] The intravenous injection of hypotonic solution is reported[3, 37] to be helpful. On the other hand, hypertonic solutions given intravenously tend to increase the symptoms.[30]

The intensity of postspinal puncture headache is increased by bilateral jugular compression. This occurs in spite of the well known fact that this procedure is accompanied by a substantial rise in cerebrospinal fluid pressure. Jugular compression, in addition to causing a secondary rise in cerebrospinal fluid pressure, results in an earlier primary rise in intracranial venous pressure. This increase in symptoms with jugular compression undoubtedly is the result of stimulation of the pain-sensitive areas by distention of veins and perhaps by an increase in volume of the brain. This increase in symptoms from jugular compression is difficult to reconcile with the benefits derived from the use of tight abdominal binders, reported by Weintraub, Antine and Raphael.[40]

It seems likely that postspinal puncture headache is caused by the stimulation of the pain-sensitive areas in the cranium that are concerned with anchoring the brain to the cranial vault. The chain of events leading to stimulation of these pain-sensitive areas may be as follows: (1) lowering of the cerebrospinal fluid pressure due to decreased volume, (2) production of a greater differential between the cerebrospinal fluid pressure and the intracranial venous pressure, bringing about (3) dilation of the venous structures and perhaps some increase in brain volume because of the venous dilation and edema.

What are the characteristic features of drainage or postspinal puncture headache? The headache occurs as a sequel to spinal puncture at varying intervals, from a few hours to several days. The headache may be mild but is frequently a dull, deep ache. It is usually not, but on occasions may be, throbbing. It is more often frontal but may be occipital, suboccipital or bitemporal. A small proportion of patients complain of pain or stiffness at the nape of the neck.

Characteristics of postspinal puncture headache which would seem to indicate that it is due to stimulation of the pain-sensitive areas as a result of lowering of cerebrospinal fluid pressure are: (1) the headache is relieved by intraspinal injection of physiologic saline solution in amounts sufficient to restore spinal fluid volume, (2) the headache is more severe when the patient is in the erect position, (3) it is usually relieved or made much milder when the horizontal position is assumed, (4) shaking of the head increases the severity of the headache and (5) the headache is made worse by jugular compression.

PREVENTION

Although it is difficult to evaluate the benefits of each measure designed to decrease the incidence of postspinal puncture headache, other than the obvious one of using some other kind of anesthesia, the adoption of certain measures would seem reasonable, even though it cannot be said that strict adherence to any one or all of them will prevent postspinal puncture headache.

It is well known that anyone's reaction to discomfort related to the head is in no way different from his reaction to pain in other parts of the body. It is probably wise to select some other form of anesthesia for patients who have a history of severe headaches or who are obviously likely to react poorly to pain of any type. Furthermore, except for very unusual reasons, spinal anesthesia should not be selected for patients who have a history of headache following a previous spinal puncture. However, the authors' investigations indicated that the history of a headache following spinal anesthesia does not mean that subsequent spinal anesthesia will necessarily be followed by a headache. Conversely, freedom from headache after one spinal puncture is not a guarantee of permanent immunity.

Anything that tends to reduce the leakage of spinal fluid from the subarachnoid space after spinal puncture would be expected to decrease the incidence of spinal puncture headache. There has been a tendency to use smaller and smaller spinal puncture needles. Cann and Wycoff[9] reported upon a series in which a 27 gauge needle was used for spinal anesthesia in an attempt to reduce the number of cases of postspinal puncture headache. The incidence of headaches with this small needle was approximately 5 per cent. Greene[15] (1949) reported a decrease in the incidence of headache following spinal anesthesia employed for vaginal delivery when he changed from a 22 gauge to a 24 gauge needle. More recently[14] (1950) he advocated the use of a 26 gauge needle and noted a decrease in the incidence of postspinal headache from 22 per cent to 0.4 per cent in 700 patients. Recently Whitacre[41] advocated the use of a needle with a point resembling that of a pencil. This needle is designed to separate dural fibers rather than sever them. He reported a significant decrease in the incidence of postspinal headache when this needle was used. Maxson[31] suggested that the bevel of the needle should be parallel with the long axis of the body so that there will be a tendency to separate fibers of the dura rather than to cut them in two. Franksson and Gordh[12] counted dural fibers severed with the needle point and noted fewer fibers cut when the bevel was held parallel to the long axis of the patient.

If the patient is held very quiet during the spinal puncture the danger of a dural tear is probably lessened. The approximately 50 per cent greater incidence of headache in the present series when the continuous technique was employed (Table 1) would seem to lend support to this assumption. It seems likely that movements of the vertebral column incidental to turning the patient into position with a needle in the subdural space would tend to enlarge the dural opening. Furthermore, an increased incidence of postpuncture headache might be expected if more than one puncture is made.

The insistence that patients be kept in the horizontal position without a pillow for a given length of time after operation probably is of little value in the prevention of postpuncture headaches.[1] Apparently the patient who will develop a headache following spinal puncture will do so irrespective of whether or not he is kept flat in bed for 24 to 48 hours following spinal puncture. This is understandable in light of the long time the opening made in the dura by the spinal needle remains patent.

Kaplan and Arrowood[22] reported a significant decrease in the incidence of postspinal headache when they injected 10 to 20 cc. of physiologic saline solution into the epidural space immediately following the injection of the anesthetic agent. After the agent was injected they merely withdrew the spinal puncture needle until the point was in the epidural space, then injected the saline solution before removing the needle. They explained this benefit on the theory that a head of pressure in the epidural spaces prevents leakage until the hole can be sealed by a fibrin clot or by the pia-arachnoid.

Increased fluid intake following spinal puncture might be expected to be of both prophylactic and therapeutic value. Recently the authors instituted the practice of administering intravenously 1,000 cc. of

5 per cent dextrose in water to all patients receiving spinal anesthesia except when such a procedure is contraindicated for some medical reason. This is started in the operating room and is done regardless of how minor the surgical procedure. Although sufficient data are not as yet available, the impression thus far is that the incidence of postspinal puncture headaches has been materially decreased since this regimen has been followed.

TREATMENT OF POSTPUNCTURE HEADACHE

The treatment of this most distressing complication still leaves much to be desired. Fortunately, most postspinal puncture headaches are mild and respond well to ordinary analgesics, such as aspirin.

Pituitrin has been used both prophylactically and therapeutically for postspinal puncture headache, probably with the idea of decreasing fluid excretion, but its value is questionable.[3, 15, 32, 33, 37] Caffeine sodium benzoate has been used more or less empirically for years. The effectiveness of this drug is likewise doubtful.

Deutsch[11] reported encouraging results following intravenous infusion of 5 per cent ethyl alcohol in 5 per cent dextrose in distilled water. A total of 1,000 cc. of solution was given in three and a half to four hours. Deutsch sometimes found it necessary to give a second infusion. This treatment is aimed at dilatation of the vessels of the choroid plexus and at the same time supplying a hypotonic solution to enter into the formation of cerebrospinal fluid.

Krueger, Stoelting and Graf[26] used 500 to 1,000 cc. of 5 per cent dextrose in .45 per cent sodium chloride to which was added 100 mg. of nicotinic acid, given intravenously, on the same basis, also with beneficial results.

Targowla and Lamache,[38] in 1927, mentioned the use of ergotamine in the treatment of spinal puncture headaches. Guttman[16] reported the drug gave complete relief in 82 per cent of patients with postpuncture headaches. Lennox, von Storch and Solomon,[27] however, stated that it was of no value in the treatment of drainage headache.

True postspinal puncture or drainage headache can always be greatly relieved and usually completely eliminated by placing the patient in the horizontal position. This may be quite objectionable to a patient who has had a spinal anesthetic for a relatively minor surgical procedure and, except for the headache, has little if any discomfort. Sometimes it may be helpful to resume the erect position gradually once the headache has been relieved by assuming the horizontal position. To do this, the bed can be turned up in stages, with several minutes or even hours taken to change from horizontal to completely erect.

The more intractable postspinal puncture headache should be investigated carefully. A lumbar puncture should be performed and the pressure of cerebrospinal fluid determined. Chemical, microscopic and bacteriologic investigations should be carried out. This is particularly applicable in cases of persistent headache unaffected by postural changes.

As with experimental drainage headache, postspinal puncture headache can always be relieved by injection of physiologic saline solution to restore the pressure of the cerebrospinal fluid. To subject a patient with postspinal puncture headache to another spinal puncture requires courage both on the part of the patient and of the physician. Although it would seem that only transitory relief might be expected from the restoration of spinal fluid pressure to normal by the subarachnoid injection of physiologic saline solution, this relief may be permanent after a single injection. If it is not, the procedure may be repeated and the relief may be permanent after the second or third injection.

Rice and Dabbs[35] reported that by peridural injections of saline solution they obtained relief of postpuncture headache in 21 of 22 patients. They demonstrated that the epidural injection of saline solution produced a prompt rise of as much as 100 mm. of pressure in the subarachnoid space. The rise in cerebrospinal fluid pressure and relief of headache was attributed to a "splinting" effect of the epidural fluid. From observations in a few cases in which the authors have used this method it seems to be very worth while. It has the obvious advantage over subarachnoid injection of saline solution of not requiring a second puncturing of the dura.

The use of abdominal binders for the relief of spinal puncture headache may be helpful particularly if the headache has developed after the use of spinal anesthesia for delivery.[40]

REFERENCES

1. Adler, H.: Study of headaches following diagnostic spinal taps, N. Y. State J. Med., 43:1328-1330, July 15, 1943.
2. Ahearn, R. E.: Management of severe postlumbar puncture headache, N. Y. State J. Med., 48:1495-1498, July 1, 1948.
3. Alpers, B. J.: Lumbar puncture headache, Arch. Neurol. and Psychiat., 14:806-812, Dec. 1925.
4. Andros, G. J., and others: Spinal (saddle block) anesthesia in obstetrics, Am. J. Obst. and Gynecol., 55:806-820, May 1948.
5. Babcock, W. W.: The dangers and disadvantages of spinal anesthesia, N. Y. State J. Med., 98:897-903, 1913.
6. Backer-Gröndahl, N.: Findings in the spinal fluid after spinal anesthesia, Acta. chir. Scandinav., 74:448, 1934.
7. Batson, O. V.: Function of vertebral veins and their role in spread of metastases, Ann. Surg., 112:138-149, July 1940.
8. Black, M. G.: Spinal fluid findings in spinal anesthesia, Anes., 8:382-389, July 1947.
9. Cann, J. E., and Wycoff, C. C.: Incidence of headache

with use of 27 gauge spinal needle, Anes., 11:294-298, May 1950.

10. Cullen, W. G., and Griffith, H. R.: Postpartum results of spinal anesthesia in obstetrics, Anes. and Analg., 26:114-121, May-June, 1947.

11. Deutsch, E. V.: The treatment of postspinal headache with intravenous ethanol: a preliminary report, Anes., 13:496-500, Sept. 1952.

12. Franksson, C., and Gordh, T.: Headache after spinal anesthesia and technique for lessening its frequency, Acta chir. Scandinav., 94:443-454, 1946.

13. Greene, H. M.: Technic to reduce incidence of headache following lumbar puncture in ambulatory patients with plea for more frequent examination of cerebrospinal fluids, Northwest Med., 22:240-245, July 1923.

14. Greene, B. A.: A 26 gauge lumbar puncture needle: Its value in the prophylaxis of headache following spinal analgesia for vaginal delivery, Anes., 11:464-469, July 1950.

15. Greene, B. A., Goldsmith, M., and Lichtig, S.: The prevention of headache after spinal analgesia for vaginal delivery by the use of hydration and a 24 gauge needle, Am. J. Obst. and Gynecol., 58:709-717, Oct. 1949.

16. Guttman, S. A.: The treatment of post-lumbar-puncture headaches with ergotamine tartrate, Arch. Neurol. and Psychiat., 49:556-558, April 1943.

17. Hand, L. V.: Personal communication.

18. Heldt, T. J., and Moloney, J. C.: Negative pressure in epidural space; preliminary studies, Am. J. Med. Sci., 175: 371-376, March 1928.

19. Hingson, R. A., Ferguson, C. H., and Palmer, L. A.: Advances in spinal anesthesia, Ann. Surg., 118:971-981, Dec. 1943.

20. Jacobaeus, H. C., and Frumerie, K.: Danger of leakage of cerebrospinal fluid after lumbar puncture and its treatment, Acta med. Scandinav., 58:102-108, 1923.

21. Jennings, W. K., and Karabin, J. E.: Incidence of headache, nausea and vomiting following spinal anesthesia, Am. J. Surg., 46:317-320, Nov. 1939.

22. Kaplan, M. S., and Arrowood, J. G.: Prevention of headache following spinal anesthesia; the use of epidural saline: a preliminary report, Anes., 13:103-107, Jan. 1952.

23. Kennedy, A. J.: Spinal anesthesia. Clinical experiences in 430 cases and technique to eliminate postoperative headache, Med. J. Australia, 1:40-45, Jan. 9, 1932.

24. Konwaler, B. E.: Changes in the cerebrospinal fluid following spinal anesthesia, Am. J. Clin. Path., 13:378-380, July 1934.

25. Koster, H., and Weintrob, M.: Complications of spinal anesthesia, Am. J. Surg., 8:1165-1179, June 1930.

26. Krueger, J. E., Stoelting, V. K., and Graf., J. P.: Etiology and treatment of postspinal headache, Anes., 12: 477-485, July 1951.

27. Lennox, W. G., von Storch, T. J. C., and Solomon, P.: The effect of ergotamin tartrate on non-migrainous headaches, Am. J. Med. Sci., 192:57-61, July 1936.

28. MacRobert, R. J.: The cause of lumbar puncture headache, J.A.M.A., 70:1350, May 11, 1918.

29. Masserman, J. H.: Cerebrospinal hydrodynamics: clinical experimental studies, Arch. Neurol. and Psychiat., 32: 523-553, Sept. 1934.

30. Masserman, J. H.: Cerebrospinal hydrodynamics; effects of intravenous injection of hypertonic solution of dextrose, Arch. Neurol. and Psychiat., 35:296-303, Feb. 1936.

31. Maxson, L. H.: Spinal Anesthesia, Philadelphia, J. B. Lippincott Co., 1938, p. 263.

32. Mixter, W. J.: Discussion, Arch. Neurol. and Psychiat., 28:959, Oct. 1932.

33. Nelson, M. O.: Postpuncture headaches, Arch. Dermatol. and Syph., 21:615-627, April 1930.

34. Pool, J. L.: Myeloscopy; intraspinal endoscopy, Surgery, 11:169-182, Feb. 1942.

35. Rice, G. G., and Dabbs, C. H.: The use of peridural and subarachnoid injections of saline solution in the treatment of severe postspinal headache, Anes., 11:17-23, Jan. 1950.

36. Sheppe, W. M.: Relation of negative pressure in the epidural space to postpuncture headache, Am. J. Med. Sci., 188:247-252, Aug. 1934.

37. Solomon, H. C.: Raising cerebrospinal fluid pressure with especial regard to effect on lumbar puncture headache, J. A. M. A., 82:1512-1515, May 10, 1924.

38. Targowla, R., and Lamache, A.: Quoted by Guttman, S. A. (reference 16).

39. Underwood, L. J.: Lumbar puncture headache; statistical analysis of 500 punctures, Am. J. Syph., Gonor. and Ven. Dis., 30:264-271, May 1946.

40. Weintraub, F., Antine, W., and Raphael, A. J.: Postpartum headache after low spinal anesthesia in vaginal delivery and its treatment, Am. J. Obstet. and Gynec., 54: 682-686, Oct. 1947.

41. Whitacre, R. J., and Hart, J. R.: Personal communication.

42. Wolff, H. G.: Headache and Other Head Pain, Oxford University Press, New York, 1948.

43. Woodbridge, P. D.: Metycaine spinal anesthesia. Report of 1381 cases, Am. J. Surg., 37:191-204, Aug. 1937.